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MORPHOLOGIC STUDIES

Smooth Muscle Contraction Bands in the Media of Coronary Arteries: A Postmortem Marker of Antemortem Coronary Spasm?

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To date, no unequivocal morphologic markers have been described that would allow the diagnosis of coronary artery spasm to be made at autopsy. The coronary arteries of 63 adult patients without myocardial infarction were examined at autopsy, and the presence of medial smooth muscle contraction bands in these vessels was correlated with other vascular changes, myocardial pathologic changes and clinical history. These contraction bands have not been reported previously in human coronary arteries, but they were identified in experimental vascular spasm induced with catecholamines. It was found that 47 of the 63 cases were positive for contraction bands. As evidence of an antemortem process, there was a significant correlation between these changes and the presence of nonocclusive microthrombi, found in 25 cases. Contraction bands were also highly correlated with atherosclerotic plaque ruptures and mural plaque hemorrhages, which may be secondary to coronary spasm. In 78.7% of the cases positive for contraction bands, the cause of death was related to a diagnosis possibly associated with high catecholamine levels. On the basis of experimental evidence and the correlations identified in this study, coronary artery medial smooth muscle contraction bands may represent a postmortem marker of antemortem coronary spasm.

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Coronary artery spasm has been documented to be a primary cause of transient coronary obstruction in patients with atypical angina pectoris (1,2) and acute myocardial infarction (3–5). In patients with acute transmural infarction, spasm may initiate the development of complete coronary occlusion, either directly or by causing mural atherosclerotic plaque hemorrhage or rupture that may lead to subsequent thrombosis (6,7). In all of these situations, the diagnosis of coronary spasm depends on the elucidation of certain clinical variables (transient ST segment elevation, relief of symptoms by vasodilating drugs) during the acute episode, or the mimicking of symptoms by a controlled clinical trial (ergonovine or cold pressor test) at later times (8,9). Ultimately, the diagnosis can be confirmed by visualization of transient constriction during coronary angiography (10). In the absence of these observations, the diagnosis of coronary spasm is speculative. In those patients who die of a coronary (or noncoronary) cause, there is no specific morphologic abnormality in the coronary vessels that allows a pathologist to conclude definitively that coronary spasm existed during life.

For some years, one of us (S.M.F.) has observed focal alterations in coronary artery medial smooth muscle cells studied at autopsy from patients dying with presumptive coronary spasm. These patients had evidence of reperfusion injury in the myocardium (contraction band or myocytolytic necrosis, or both) or plaque hemorrhage and rupture associated with luminal coronary thrombosis and myocardial infarction. The observed medial smooth muscle changes resembled contraction bands; although not generally appreciated, smooth muscle cells may undergo "hypercontraction" and give rise to dense eosinophilic bands like those in reperfused cardiac muscle (11). In vessels, contraction bands have been demonstrated in the setting of experimentally induced vasoconstriction, either in vitro (12) or in vivo (13); however, to date, these changes have not been reported in the clinical setting.

Despite the fact that we observed contraction bands in cases clinically or pathologically consistent with coronary artery spasm, we could not be certain that these alterations were not sectioning artifacts or related to postmortem rigor mortis. Accordingly, we designed the present study to evaluate the frequency of coronary contraction bands in routine autopsy tissue, and to correlate their presence or absence

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with any clinical or pathologic features that might suggest the development of coronary spasm. During the course of this study, we unexpectedly observed the frequent occurrence of nonocclusive microthrombi in the affected coronary tree, as well as plaque hemorrhages and rupture, all of which appear to be significantly associated with the contraction band process. Together, these lesions may be important morphologic markers of antemortem coronary events.

Methods

Patient selection. We elected to analyze coronary arteries in autopsied patients without acute myocardial infarction to determine the general frequency of lesions in a group of patients not dying with clinically recognized coronary artery spasm. This survey could then provide a baseline to determine whether coronary artery lesions in patients with spasm represented real or artifactual alterations. Accordingly, all adults over the age of 18 years coming to autopsy during approximately a 6 month period were entered into this study, and the only patients specifically excluded were those with a clinical diagnosis of acute myocardial infarction. In addition, a small number of patients were not included because of prosector oversight, inappropriate selection of coronary rings or inadequate histologic coronary sections.

Study cases. A total of 63 cases were evaluated in which the primary diagnosis was other than acute myocardial infarction. The mean age of the entire group was 63.9 years (range 19 to 91). There were 32 men and 31 women. Twenty-eight patients were clinically diagnosed to be hypertensive, and 35 were normotensive. These cases were considered to be representative of our usual autopsy population. During the period of this study, eight adult patients were eliminated because of a diagnosis of acute myocardial infarction. Six patients with miscellaneous diagnoses (heat stroke, intracerebral hemorrhage, lung cancer, leukemia, bowel infarction and acute pulmonary embolism) were not included for technical reasons. Eleven patients were withdrawn from the study because there were insufficient or inadequate coronary sections available for evaluation.

Coronary artery selection and evaluation. To prevent any overt bias that might result from sectioning areas with grossly evident lesions, and to sample the coronary arteries in a manner relevant to the routine pathologic evaluations performed in most autopsies, prosectors were instructed to harvest 3 to 5 mm long coronary rings at seven specified points in each case: 1) the left main coronary artery halfway between the ostium and bifurcation; 2) the mid left circumflex artery; 3) the proximal and distal left anterior descending artery; and 4) the proximal, mid and distal right coronary artery. In cases with extensive severe obstructive coronary artery disease, sections were also removed from the involved areas away from the seven designated points. Each ring was labeled and fixed in 3.7% buffered formaldehyde. If the vessel was markedly atherosclerotic, the coronary ring was decalcified before sectioning. Each coronary segment was separately processed, embedded in paraffin, cross-sectioned at three to five levels perpendicular to its lumen and stained with hematoxylin-eosin. The vessels were examined individually by both authors who evaluated the variables described below. Any discrepancy between the two evaluations was resolved by joint consultation (in 5 out of 63 cases), and a final decision was reached.

The seven coronary rings from each case were examined without any knowledge of the clinical history or the gross and microscopic myocardial examination. Sections that were disrupted during processing, or cut tangentially, were not evaluated; any case with more than two such vessels was eliminated from the study. Each ring was visually circled, and an estimate was provided for the percent luminal occlusion by atherosclerosis. The entire vessel wall was examined at scanning and high power objectives to evaluate medial smooth muscle alterations. The presence or absence of contraction bands was noted; vessels with fewer than three such lesions or with questionable lesions were considered to be negative. This exclusion was employed to eliminate the possibility that a staining or other unusual histologic artifact might be misinterpreted as a contraction band. Contraction bands were defined as discrete areas of hypereosinophilic density, usually associated with transverse widening of the cell diameter and rarefaction of the cytoplasm peripheral to the band. When contraction bands were identified, attention was paid as to whether they were circumferential or focal. If focal, their presence in relation to intimal atherosclerotic plaques was recorded (either directly beneath a plaque or on the opposite vessel wall).

Other medial smooth muscle alterations were evaluated both in relation to contraction bands and as they occurred generally throughout the vessel wall. These changes included evidence of nuclear contraction or shortening, with marked folding of the nuclear membrane and condensation (or increased density) of the nuclear chromatin. The presence of inflammation associated with contracted smooth muscle cells, and vacuolization of cells throughout the media were also observed. Finally, three luminal or mural abnormalities were evaluated for each vessel segment: the presence of atherosclerotic plaque hemorrhage, plaque rupture or disruption, or both, and luminal thrombosis.

Clinical data. A number of clinical variables were tabulated for each case without regard to the pathologic findings in the coronary arteries and myocardium. Features noted included patient age, sex, blood pressure (both history of hypertension and in-hospital blood pressure during the terminal illness), clinical presentation and mode of death (sudden and unexpected, or chronic and expected) if entered in the records. If the pathologic diagnosis determined at postmortem examination differed from the clinical diagnosis,

the former was used in this study. Finally, to establish whether rigor mortis was related to the presence of contraction bands in the coronary arteries, the approximate time from death to autopsy was noted.

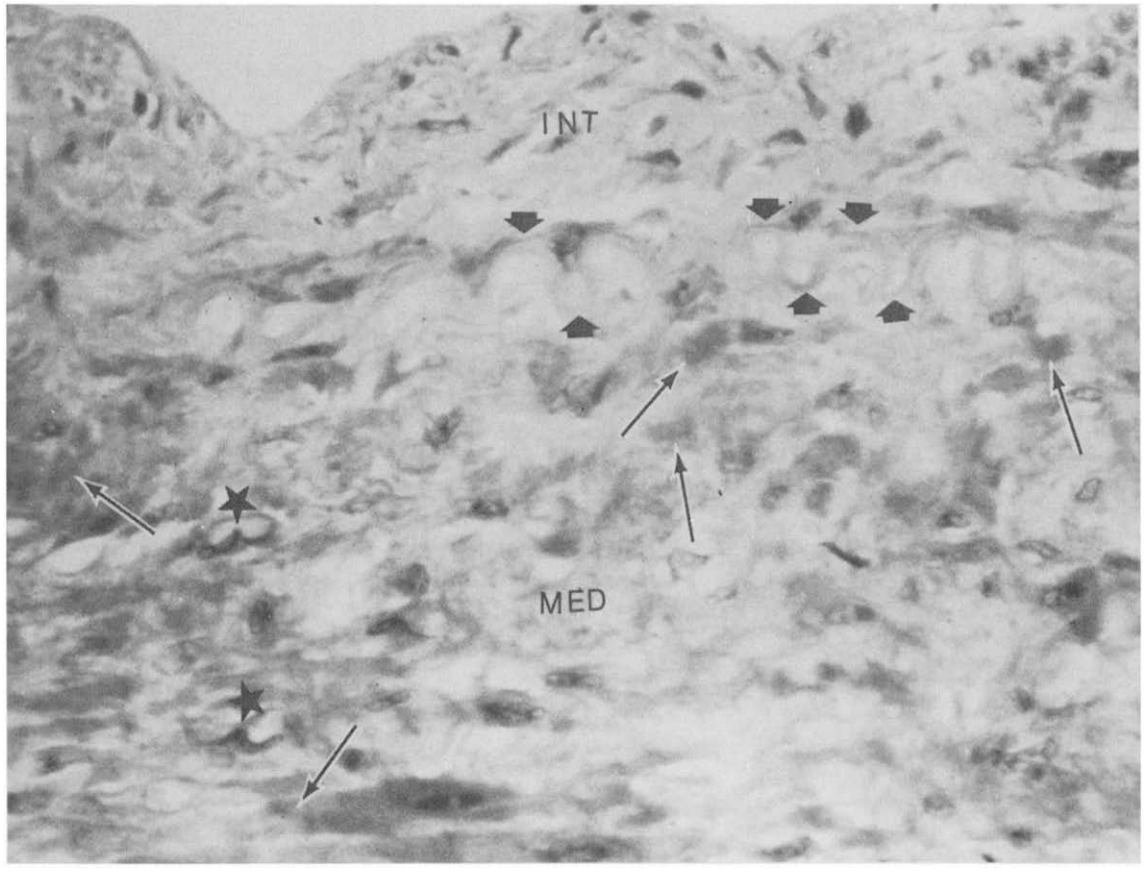
Statistics. All statistical analyses and correlations were performed by chi-square tests. A probability (p) value less than 0.05 was considered significant.

Results

Coronary artery contraction bands. Forty-seven (74.6%) of the 63 cases were found to be positive for contraction bands (Fig. 1 and 2). A total of 391 vessel segments (not including multiple levels) were examined from the 63 cases (mean 6.2 vessels/case); of these, 162 (41.4%) were positive. Contraction bands were located circumferentially through the media of the vessel wall in 80.4% of the cases, while 11.1% were present opposite atherosclerotic plaques and 8.5% were found above or just lateral to plaques. Several vessels in which contraction bands were common had a folded or contracted appearance or had prominent splitting or separation of the vascular layers. These features were not quantitated because of our uncertainty in differentiating them from sectioning artifacts; however, they were rarely found in vessels without contraction bands. Of note, contraction bands were more common in vessels with less than 50% occlusion by atherosclerotic plaque (62.2%) than in vessels with greater than 50% occlusion (37.8%). When we analyzed vascular involvement on a case by case basis, we observed that the presence of contraction bands was not a generalized phenomenon (that is, they were not found in all four vessels from any one case), but they were often localized. In 13 cases (27.7%), they were present in only one vessel, in 13 they were identified in two vessels and in another 13 they were seen in three vessels. In only eight cases (17.0%) were contraction bands found in all four vessels.

Analysis of other changes in the coronary media associated with contraction bands revealed that smooth muscle nuclear folding or condensation (Fig. 1) was found in 37 cases (79.6%) compared with 5 cases (31.3%) of the 16 without contraction bands ($p < 0.01$). Inflammatory cell infiltrates, which were predominantly round cells not as-

Figure 1. A section of a widely patent distal left anterior descending coronary artery from a 68 year old woman with metastatic cancer treated with adriamycin and found dead. The intima (INT) is unremarkable. The media (MED) is disorganized and vacuolated. Numerous smooth muscle contraction bands are present, some of which have been delineated with **arrows**. The internal elastic lamina (**arrowheads**) appears markedly folded. Several smooth muscle nuclei (**starred**) also are contracted. (Hematoxylin-eosin, magnification ×480, reduced by 10%.)



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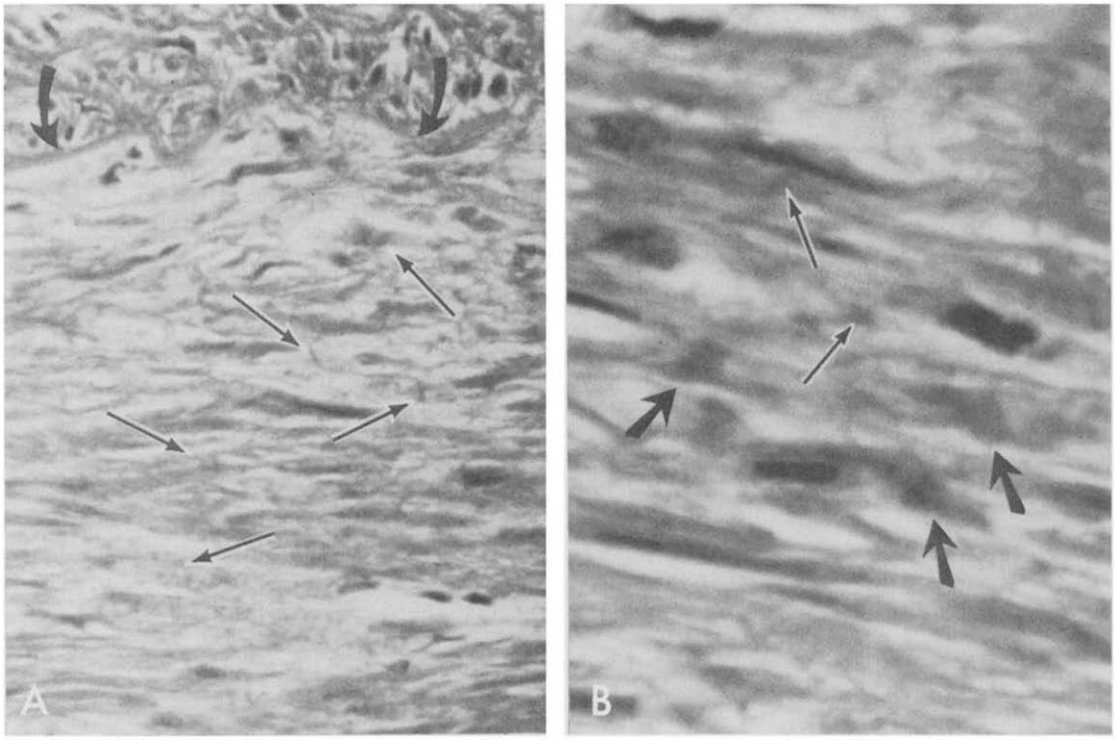

Figure 2. Sections of proximal left anterior descending coronary artery with 60 to 70% luminal narrowing. **A**, Multiple small and large contraction bands (**thin arrows**) are seen in virtually every smooth muscle cell in this region of the vessel opposite an atherosclerotic plaque. The internal elastic lamina (**curved arrows**) is unremarkable. The patient was 85 years old and was found dead with no apparent cause. (Hematoxylin-eosin, magnification ×480, reduced by 10%.) **B**, A different area of the media from the same vessel. At high magnification, a number of large dense smooth muscle contraction bands (**thick arrows**) can be seen along with multiple small regions of cytoplasmic condensation (**thin arrows**). (Hematoxylin-eosin, magnification × 1,200, reduced by 10%.)

sociated with intimal or adventitial inflammation, were found adjacent to contraction band changes in 16 positive cases (34.0%). In three negative cases (18.8%), they were also observed in the media (p = NS). We did not find vacuolization of medial smooth muscle cells to be a useful diagnostic sign because vacuoles were observed in virtually every positive and negative case.

Coronary artery microthrombi. The most interesting observations were related to the association of coronary contraction bands with vessel wall or luminal alterations and myocardial lesions (Table 1). Twenty-five (53.2%) of the 47 cases with contraction bands had nonocclusive thrombi predominantly adherent to the intimal surface, while a few were loose within the vascular lumen. Most of the thrombi were of microscopic dimensions and composed of granular, eosinophilic material consistent with platelets (Fig. 3 to 5). A few microthrombi were associated with fibrin or cellular elements including red cells and leukocytes organized compactly or loosely along the intimal surface. Several microthrombi were identified in which the underlying endothelial cells were either sloughed or organized into the thrombus. Although most microthrombi appeared to be recently formed, a small number were covered by endothelial cells and already incorporated into the intima. Quantitative analysis revealed that in the 25 cases microthrombi occurred in 44 (95.7%) vessel segments with concurrent contraction bands, while microthrombi were observed in just 2 (4.3%) vessel segments without contraction bands. Sixty percent of microthrombi were found in vessels with less than 50% atherosclerotic occlusion. In the 13 cases in which contraction bands were present in a single vessel only, seven of those vessels also had microthrombi. In the 16 cases without contraction bands, microthrombus was identified in only 1 case (6.3%), representing a highly significant ($p < 0.001$) difference from the positive cases.

Coronary artery plaque rupture and hemorrhage. In 5 (10.6%) of the 47 cases with contraction bands, complete or incomplete plaque rupture or disruption was observed, with release of plaque contents into the vascular lumen. Overlying nonocclusive thrombus was noted in one such case; in another case not tabulated as positive, a fresh platelet microthrombus contained a hemosiderin-laden macrophage, presumably from a plaque rupture that was not found in the random vessel segments chosen for study. No plaque rupture was identified in any of the 16 cases without contraction bands. Mural plaque hemorrhages without plaque rupture were found in 15 cases (31.9%) with contraction bands, and in only 2 cases (12.5%) without contraction

Table 1. Pathologic Features of 63 Cases

Contraction Bands (no. of cases)	Microthrombi	Nuclear Changes	Medial Inflammation	Plaque Rupture	Plaque Hemorrhage
47 (+)	25 (53%)	37 (79%)	16 (34%)	5 (11%)	15 (32%)
16 (−)	1 (6%)	5 (31%)	3 (19%)	0 (0%)	2 (13%)

+ = positive; − = negative.

bands. Although the association between either plaque rupture or mural hemorrhage and medial contraction bands clearly suggested a positive trend, the analysis failed to reach statistical significance at the 0.05 level because of the relatively small number of cases involved.

Acute myocardial lesions. The myocardium from the 63 cases was analyzed for the presence of acute lesions. Thirty-six of the 47 cases with coronary contraction bands had microscopic foci of acute myocardial contraction band necrosis or early myocytolysis. These myocardial changes were also observed in 6 of the 16 cases without coronary contraction bands. The association of coronary and myocardial lesions was significant at the $p < 0.01$ level.

Clinical associations. The clinical features of the 63 cases are summarized in Tables 2 and 3. No significant differences were noted in the age and sex distribution of

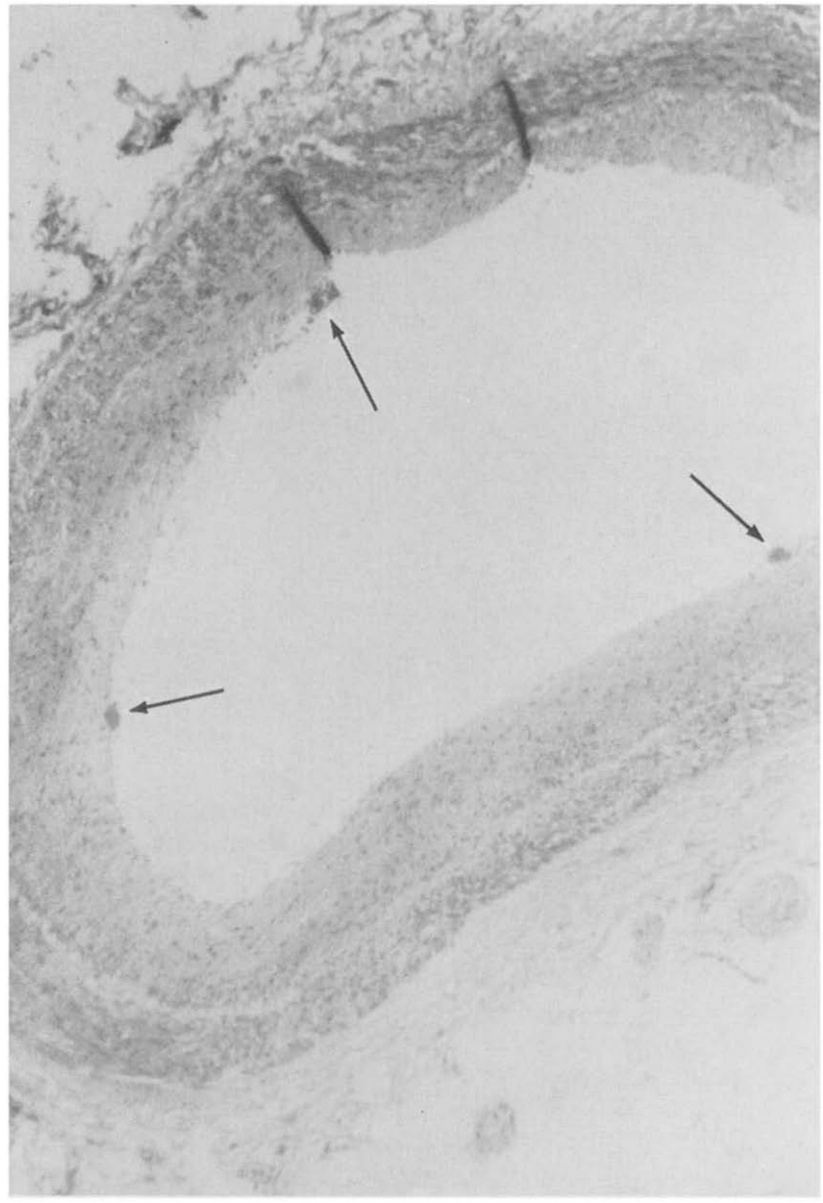

Figure 3. This widely patent vessel (proximal left anterior descending coronary artery) was from a 58 year old man who had cerebral coma due to meningitis. The medial smooth muscle cells have diffuse contraction band changes that cannot be seen at this magnification. Three microthrombi (**arrows**) are adherent to the vessel wall, with no obvious cause other than their highly significant association with medial contraction bands. (Hematoxylin-eosin, magnification ×75, reduced by 10%.)

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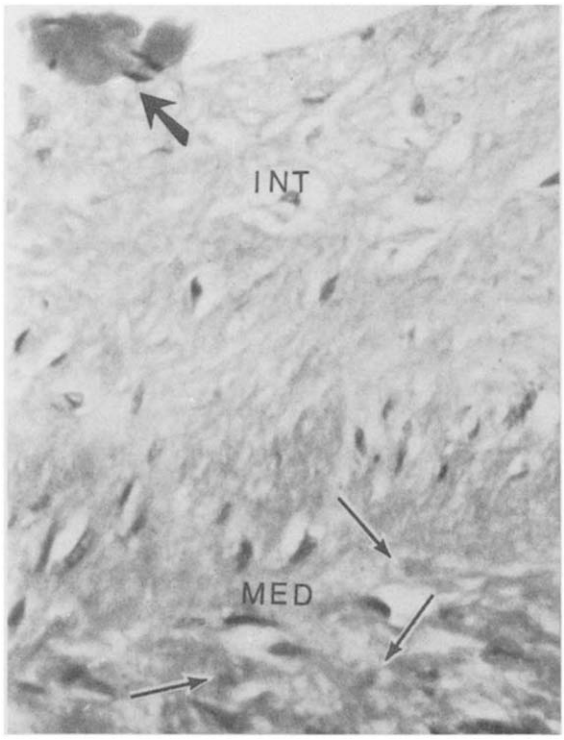

Figure 4. Two microthrombi (**thick arrows**) from a different segment of the vessel seen in Figure 3. The thrombi are present in an area of endothelial sloughing and are partially adherent to intimal connective tissue. Two condensed endothelial cells (**thin arrows**) appear to be loosely attached to one microthrombus. The subendothelial connective tissue is markedly edematous (**star**). (Hematoxylin-eosin, magnification ×480, reduced by 10%.)

patients or the presence or absence of hypertension in either group. When the time from death to autopsy was categorized into three groups (< 12, 12 to 24 and > 24 hours), no statistically significant relation was found between this time and contraction band changes in the coronary arteries. Retrospective analysis of the primary causes of death (Table 3) reveals that 78.7% of the cases with positive contraction bands had a diagnosis of shock (hemorrhagic or septic, n = 14), cardiomyopathy (alcoholic, amyloid or adriamycin, n = 5), central nervous system lesion (hemorrhagic stroke, ruptured aneurysm or cerebral mass, n = 10) and sudden death (arrhythmia, pulmonary embolism or unexplained, n = 8). Similar analysis revealed that 31.3% of the cases without contraction bands had a diagnosis of shock (septic, n = 2), cardiomyopathy (alcoholic, n = 1), central nervous system lesion (brain tumor, n = 1) or sudden death (pulmonary embolism, n = 1). When these four diagnostic categories were combined, statistical analysis revealed a highly significant ($p < 0.001$) association with the presence of contraction bands. In both positive and negative cases, some patients had two or more diagnoses which were sufficiently severe to cause death. In these instances, we chose the most likely cause; however, this selection process was subjective.

Discussion

The present report describes the presence of contraction bands in the media of coronary arteries from a nonselected group of patients dying without myocardial infarction. To our surprise, we found these lesions in 47 (74.6%) of our 63 cases; but rather than suggesting that these changes are artifacts, we believe that their association with nonocclusive coronary artery microthrombi and atherosclerotic plaque hemorrhage or rupture, or both, strongly suggests that coronary smooth muscle contraction bands are markers of in vivo phenomena consistent with coronary artery spasm. If this interpretation is correct, then the prevalence of such lesions even in "noncoronary" cases implies that coronary artery spasm possibly may play a significant role in the terminal course of many diverse diseases.

Previous studies. To date, no definite pathologic changes at autopsy that can identify coronary artery spasm occurring during life have been described. There have been several

Figure 5. A segment of a distal right coronary artery with 10 to 20% luminal narrowing from a 50 year old woman who died suddenly after an acute cerebral hemorrhage. A microthrombus (**thick arrow**), in which several probable endothelial cells have been incorporated, is adherent to the intimal (INT) connective tissue. Within the media (MED) underlying the microthrombus, numerous smooth muscle cells contain contraction bands (**thin arrows**). (Hematoxylin-eosin, magnification ×480, reduced by 10%.)

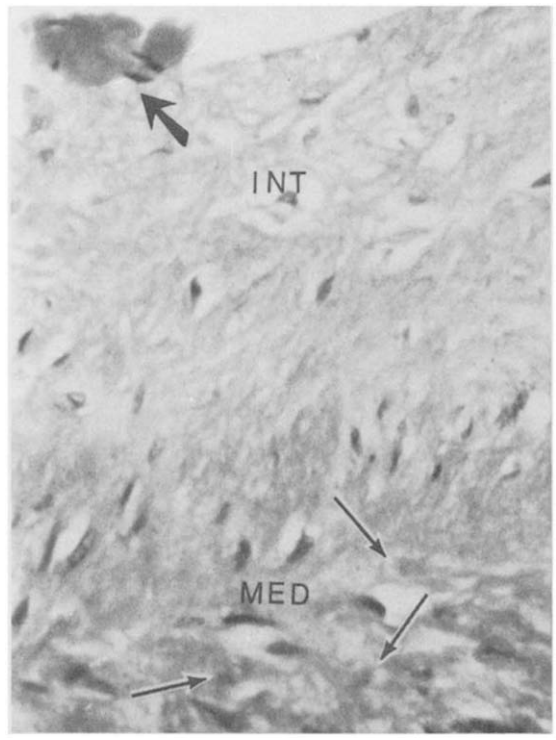

Table 2. Clinical Features of 63 Cases

Contraction Bands (no. of cases)	Mean Age (yr)	Sex		Hypertension/ Normotension	Hours Postmortem		
		M	F		12	12 to 24	24
47 (+)	63.1	24	23	22/25	10 (21%)	23 (49%)	14 (30%)
16 (−)	71.6	8	8	6/10	2 (12%)	10 (63%)	4 (25%)

+ = positive; − = negative.

recent reports (14,15) in which postmortem spasm was diagnosed because of persistent coronary hypercontraction. El-Maraghi and Sealey (14) reported on a 25 year old man with a previously normal coronary angiogram, recurrent myocardial infarction and multiple grossly evident segmental constrictions in the coronary tree. Roberts et al. (15) described three patients with Prinzmetal's angina and sudden death, who had coronary spasm on angiography and focal histologically contracted coronary segments at autopsy corresponding to the angiographically spastic segments. In neither report did the authors comment on morphologic changes in the vessel media. Thus, the diagnosis of spasm in both studies was dependent on the presumably rare persistence of gross coronary hypercontraction at autopsy, and the ability to correlate postmortem alterations in the coronary tree with antemortem angiography. Because most patients coming to autopsy have not had coronary angiograms in the period immediately before death, it would be very helpful if some morphologic marker of coronary spasm could be developed for the establishment of the diagnosis postmortem.

Smooth muscle contraction bands persisting for hours after either an in vivo or in vitro pathologic insult have been described in both the muscularis propria of bowel (11) and the media of blood vessels (13). These hypereosinophilic bands are similar to those described in the myocardium over the past 20 years by numerous investigators (16,17). In the bowel, they were ascribed to the effects of ischemia or localized release of catecholamines, or both, while catecholamines have also been implicated in the development of myocardial lesions (18). Ming and McNiff (11), who illustrated the light and electron microscopic nature of these bands in intestinal muscularis, depicted zones of myofilament hypercontraction with associated cytoplasmic rarefaction peripheral to these areas, as well as nuclear shrinkage and basophilia, all features identified in the present study.

In regard to contraction bands and vascular constriction, Van Citters et al. (12) described the effects of epinephrine on freshly excised vascular segments that were treated and then rapidly frozen. These workers illustrated marked contraction of the vessel ring with associated nuclear shortening and deformation. They demonstrated that these changes could occur focally if the catecholamine was applied to only one side of the vessel. Of note, however, is that although the authors did not comment on this point, their illustrations clearly appear to demonstrate focal smooth muscle contraction bands in the constricted segments. More relevant to the present report, Joris and Majno (13) described the effects of in vivo applications of norepinephrine to vessels with subsequent pathologic study up to 12 hours later. These investigators noted several types of damage in medial smooth muscle cells from spastic vessels, including the development of persistent supercontraction and shedding of myofilaments. We believe that their characterization of these lesions represents the ultrastructural correlate of the medial contraction bands reported by us. Thus, on the basis of previous studies (12,13), it appears that in experimental vascular spasm induced by catecholamines, contraction band alterations of medial smooth muscle cells occur focally and may persist after the development of the lesion.

Table 3. Clinical Diagnosis in 63 Cases

	Contraction Bands	
	(+)	(−)
Hemorrhagic CVA/SAH	7	0
Alcoholic cardiomyopathy	2	1
Amyloid cardiomyopathy	1	0
Hemorrhagic shock	7	0
Sudden death/arrhythmia	6	0
Brain tumor/metastases	3	1
Acute pulmonary embolism	2	1
Septic shock/sepsis	7	2
Cancer (adriamycin)	8 (2)*	6
Miscellaneous	4	5
Total	47	16

*Two patients with cancer or adriamycin-induced cardiomyopathy.
CVA/SAH = stroke or primary subarachnoid hemorrhage, or both.

Artifacts or real? Because of the prevalence of contraction bands in our study, we analyzed the pathologic and clinical data carefully to determine whether there were objective criteria that might establish whether the lesions were artifactual or real. Contraction bands are likely to be present diffusely throughout the coronary tree if they are artifacts. Yet, they were found in all four of the sampled vessels in only 8 cases, while in 13 cases (27.7%) they were identified in a single vessel. In only two cases, representing 3.2% of the entire series, were they noted in every one of the seven vessel segments studied from each heart. Furthermore, there was no relation between the time from death to autopsy examination, thus suggesting that rigor mortis (which would

also be expected to affect all coronary vessels from any one heart equally) played no role in their development. Because all vessel segments were selected from the coronary tree in a similar manner, no specific bias should have been introduced.

The strongest argument that contraction bands are in vivo phenomena relates to their association with vascular changes that demonstrate objective evidence of antemortem events. In 20 cases positive for contraction bands, we found plaque ruptures or mural hemorrhages, whereas these features were noted only in 2 of 16 negative cases. Admittedly, prosectors may have selected coronary segments with these grossly evident lesions if they occurred near the specified regions to be saved; if so, this might bias our findings if these mural changes were also postmortem artifacts. However, we only scored a segment as positive for these alterations if there was evidence of an unequivocal antemortem event, such as overlying thrombus, intermixture of plaque material with fibrin and inflammatory cells or the presence of hemosiderin. The presence of medial splitting or plaque fracture, which also was associated with contraction bands, was not quantitated because it might represent a sectioning artifact.

The association of contraction band alterations with intimal nonocclusive microthrombi is of even greater importance because these lesions could not be identified grossly in most instances, thus precluding a biased selection of material. Microthrombi, predominantly composed of compact eosinophilic granular material consistent with platelets, were observed in more than 50% of the cases that were positive for contraction bands. Furthermore, in the 46 vessel segments with microthrombi, 44 had contraction bands. These microthrombi were clearly due to antemortem events, and in virtually every case where they were found they were densely adherent to the intimal surface. In several cases, they were intermixed with sloughed endothelial cells and noted on the denuded intimal surface, or they were organized and incorporated into the intima.

Coronary artery microthrombi. Coronary nonocclusive microthrombi may be more common than is generally appreciated. Haerem (19) described a significantly increased number of platelet aggregates in the epicardial coronary arteries of patients dying suddenly with coronary atherosclerosis. Aggregates also were present in patients dying with chronic coronary disease or from noncoronary causes, representing two groups that were more comparable with those in our report. Frink et al. (20) noted nonocclusive coronary thrombi in six cases of sudden cardiac death. As with those described by Haerem (19), they appeared less compact and homogeneous than most of the microthrombi in our series. A potential mechanism for the development of microthrombi was proposed by Joris and Majno (21), who described endothelial damage and associated platelet aggregation after norepinephrine-induced arterial spasm. Other workers (22) also provided supportive evidence by demonstrating that coronary constriction, even when not sufficient to reduce the rate of blood flow through a vessel, could lead to endothelial cell fragmentation and desquamation, with subsequent platelet adherence to the underlying intima and microthrombus formation.

On the basis of the histologic findings alone, we have no way of determining whether the platelet microthrombus occurred after or preceded the development of coronary spasm. As recently proposed by Hirsh et al. (23), it is possible that thromboxane release from platelets is associated with coronary spasm in unstable angina pectoris. These workers, however, could not be certain whether the release was cause or effect. In a subsequent study of patients with vasotonic angina, Robertson et al. (24) concluded that thromboxane was released during coronary vasospasm, but that it did not cause the process. It is of interest that microthrombi and contraction bands were identified in approximately 60% of vessels with less than 50% atherosclerotic luminal occlusion, suggesting that a relative increase of smooth muscle in the media might make that vessel more likely to undergo hypercontraction. Conversely, it would be more likely that severely atherosclerotic vessels might be more susceptible to thrombus formation. However, despite these observations, one still cannot be certain which process (spasm or microthrombus) occurred first.

Cause of death. Finally, it is noteworthy to correlate the presence of coronary contraction bands with the cause of death (Table 3). Although the cases in this report are representative of our usual patient population, the fact that the majority of the subjects came from a large municipal acute care hospital probably explains why so many had shock, cardiomyopathy, central nervous system lesions and sudden death. The frequency of these diagnoses in our study group may account for the seemingly high number of cases with coronary contraction bands. The common denominator in all of these conditions is the presence of elevated levels of circulating catecholamines (25–31). If the experimental studies previously cited (12,13), demonstrating the effects of catecholamines on the development of vascular spasm and medial smooth muscle contraction bands, are relevant to clinical material, then this may provide an explanation for our findings.

Conclusion. We have described distinct contraction bands occurring in the medial smooth muscle cells of coronary arteries studied at autopsy, which are positively correlated with a variety of other pathologic findings. These include the presence of nonocclusive intimal microthrombi, plaque ruptures, mural hemorrhages and terminal conditions associated with elevated levels of catecholamines. This new observation is also supported by several clinical and experimental studies demonstrating that antemortem coronary constrictions can persist in the postmortem period. Although additional studies in specific patient groups are necessary to confirm our observations, we believe that medial smooth

muscle contraction bands are markers of terminal coronary artery spasm. If this is so, then coronary artery spasm sufficient to produce vascular or myocardial damage may be more common than is generally appreciated.

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